

CORRESPONDENCE

Health risks from exposure to cadmium in soil

We were intrigued by the report and findings from Elliott *et al*¹ of overall mortality, cancer incidence, and stroke mortality in Shipham village. Their findings are similar to the conclusions we reported in 1982 after work funded by the Department of Health and Social Security.² We noted that "the failure to demonstrate any excess morbidity requiring hospital admission is reassuring for Shipham residents". We identified a small but significant excess of carcinoma of the ovary but thought it extremely unlikely that this could be explained by exposure to cadmium; the histology of the two reported neoplasms was different and one of the patients resided at an address with a normal soil cadmium content. Hospital admissions were not increased for essential benign hypertension, hypertensive heart disease, acute nephritis, other nephritis and nephrosis, and calculus of the urinary system,³ or from gastric cancer which had in North Wales been associated with similar patterns of contamination of the soil with heavy metal.⁴ These findings are not explored by Elliott *et al*. Although they explore biomarkers of blood and urine, they do not discuss the worth of *in vivo* neutron activation analysis or dental studies. Yet, in 1979, it was reported that the mean (SD) liver cadmium concentrations of 21 Shipham residents was 11.0 (2.0) ppm, which was significantly higher ($p < 0.001$) than that of 10 non-Shipham controls (2.2 (2.0) ppm). These researchers also reported values of up to 260 ppm in industrially exposed workers, and that neither the workers nor the Shipham residents showed any evidence of cadmium toxicity.⁵ These findings were considered reassuring.⁶ The dental health of Shipham children was reported to be similar to children in neighbouring villages without the soil contamination,⁷ although increased concentrations of cadmium had been found in their teeth.⁸ One other background study of the villagers, also not cited by Elliott *et al*, reported widespread morbidity among 22 of 31 village residents.⁹ Its methodology has, however, been severely criticised.¹⁰

There is no doubt that the more extensive and detailed follow up by Elliott *et al* must again be reassuring to this residential population. However, as they point out, the intervening two decades since public health studies began in this village have seen enormous changes in the composition of its residents. Moreover, the soil concentrations of cadmium within Shipham vary considerably between gardens of adjacent houses (Department of the Environment, unpublished data). Dose-response relations for the population of Shipham residents to all sources of cadmium are extremely difficult to estimate.¹¹

This public health interest in the soil concentrations of cadmium in Shipham village first arose in 1979 as a response by the United Kingdom Government Central Directorate on Environmental Pollution, Department of the Environment, to widespread news media coverage of work being undertaken by the Department of the Environment for the distribution of cadmium in the

environment.¹¹ This work followed up findings reported in the Wolfson geochemical atlas of England and Wales.¹² At that time the contamination which had been widely known for generations within the village was not considered to present any known health risks for the population.¹³ Elliott *et al* do not seem to be aware of this background or of the findings from earlier studies. Such an introduction would have helped to set the context of their own study.

The earlier publications also helped to make the study of Elliott *et al* possible by reporting the huge time costs needed for manual record linkage.¹⁴ For example, they reported that "the postcode of residence was used to identify cases from the national cancer and mortality databases, held by the United Kingdom Small Area Health Statistics Unit (SAHSU).¹ Back in 1976, the need for record linkage was discussed.¹⁵ Our study of Shipham residents "was, in part, a response to the challenge that further interest should be stimulated in the use of Hospital Activity Analysis data" (HAA).² We also reported that "for HAA purposes addresses of patients are coded by local authority districts. Health problems, however, are often restricted to much smaller geographical areas. Before we could calculate standardised admission ratios for Shipham, 451 hours of clerical work were required to identify the 201 records of HAA data for Shipham residents".¹⁴ At that time the need to produce statistics for small areas had been recognised and the then Office of Population, Censuses and Surveys was introducing a postcode system for vital statistics in England and Wales. Postcoding of hospital patient data and record linkage followed in the late 1980s. Elliott *et al* were able to use these developments.

We thought that public health fears generated for this population had been allayed by studies reported in the 1980s for their sources of exposure, dietary intake of cadmium, body burden of cadmium, morbidity, and mortality experience. What Elliott *et al* now report is further evidence from longitudinal studies. The worth of such follow up studies is considerably weakened by knowledge that on average, 10% of the population move house each year,¹⁶ and difficulties estimating total body burden of cadmium.

We think that much can be learned from the experience of studies involving this population. In particular, any such long term follow up studies should be sensitive to their public health needs. As Elliott *et al* and ourselves have noted, the methodological problems associated with interpreting findings from the use of routinely available data are considerable. It is therefore important for researchers, with the ready availability of powerful, computer based literature searching facilities and library held compendia—such as the Index Medicus—to be able to reassure readers that they have considered all the relevant background information and that their findings are being fully discussed in the context of other published work. Questions the informed reader will ask include: has a comprehensive risk assessment been undertaken? Has all the evidence been considered and is it coherent? Are there anomalies and can they be explained? What fresh insights has the study yielded? And what are the implications of the findings? In this instance we are left asking why was this recent study undertaken and what has it added to existing knowledge? Or, in other words, why has this soil been turned over again? It should be realised that in 1979, as a consequence of the

news media scare and without any public health evidence, property values in the village dropped to half their market value. They took years to recover. Accordingly, we need to remind ourselves that we have a duty of care in planning research to ensure that our efforts to better understand occupational and environmental health problems are intended for the public good.

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Author's reply—We have read with interest the comments to our paper by Philipp and Hughes. They note that we did not explore hospital admissions for several diagnoses. We have, however, used hospital admissions data in other Small Area Health Statistics Unit (SAHSU) studies, and found that analyses with such data are far from straightforward. It is doubtful that analysis of health outcomes—such as benign hypertension or calculus of the urinary system—would give any meaningful results, as most cases will not be admitted to hospital. We did not analyse incidence of gastric cancer, as there is no evidence that cadmium is a risk factor for this cancer.¹

Philipp and Hughes state that we "explored blood and urinary markers", whereas we noted that "biological data (cadmium in blood or urine) were not available for use in the present investigation". We saw no reason to "discuss the worth of *in vivo* neutron activation analysis", because such methods are not particularly useful for exposure assessment in epidemiological studies.²

We are fully aware of the historical background, which we described in the introduction to our paper, including a reference to the Wolfson geochemical atlas.³ We also referred to the original cohort analysis by Inskip *et al*, and several papers from the com-

prehensive Shipham report.^{4,7} One of us (IT) was involved in the original Shipham study, and another one of us (MQ) was closely involved in much of the research published in the Shipham report.⁸

We acknowledge the difficulties involved in small area health studies in the 1970s. We are of course grateful for all developments, including postcoded data and new computer hardware and software, that have led to a much more efficient use of routinely collected morbidity and mortality statistics, and of research resources. Researchers at SAHSU have contributed substantially to this development, for example by enabling rapid computations of risks associated with potential environmental pollution to be made.⁹

We are aware of the problems introduced by migration when analysing health effects with long latency times, and we commented on this in relation to the "geographical" study.

Our study was not intended to be a "comprehensive risk assessment"; we do, however, refer to one.¹ The availability of a further 18 years of mortality data since the publication of the paper by Inskip *et al.*¹⁰ seems to us a legitimate reason for updating their analysis. Cancer incidence data had not previously been analysed. We agree with Philipp and Hughes, that our paper most likely is reassuring to the local population, which should be for the public good.

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Non-neoplastic mortality of European workers who produce man made mineral fibres

EDITOR—The recent publication by Sali *et al.* reports "a suggestion of an increasing risk of death from non-malignant renal diseases" among rock and slag workers with employment in the early technological phase.¹ No such relation was found for glass wool workers. The 1985 follow up of the man made mineral fibre worker (MMMMF) study in the United States reported a significant increase in mortality for nephritis and nephrosis based on 56 deaths for the entire male cohort.² Sali *et al.* concluded that additional studies are warranted. We should like to point out an additional study of glass wool workers published earlier in this *Journal* dealing with nephritis or nephrosis.

The Division of Occupational Health Studies, Department of Family Medicine, Georgetown University Medical Center maintains a mortality surveillance system (MSS) on behalf of Owens Corning (OC). The MSS includes both detailed exposure information and the results of an interview survey which provides information on socio-demographic factors including education, marital status, income, drinking, and smoking.³ We used a case-control study with cases and controls derived from the MSS to investigate the question of whether there is an association between exposure to respirable glass fibre or silica and mortality from nephritis or nephrosis among workers in fibrous glass wool manufacturing facilities.⁴

Two case-control analyses were carried out, one where the cases were defined with nephritis or nephrosis as the underlying cause and one where cases were defined as those where nephritis or nephrosis is either the underlying or a contributing cause of death.

We found no consistent relation for respirable fibres or respirable silica when the analysis was based either on underlying cause only or on underlying plus contributing cause. None of the sociodemographic variables considered suggests an increased risk when considering both underlying and contributing cause. For these data, all odds ratios for respirable fibres and silica based on both underlying and contributing cause of death are below unity with the exception of the highest exposure level for silica, which is 1.04. Although these results do not prove that there is no association between nephritis or nephrosis and exposure to fibreglass or silica in the fibreglass manufacturing environment, they do not support the assertion that such an association exists.

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Boffetta and Saracci reply—Our conclusions were based on our finding on rock or slag wool workers, not on glass wool workers, a group comparable with the one studied by Chiazze *et al.* Indeed, we reported that we found no relation between mortality from non-malignant renal diseases and employment in glass wool production. Given that the other large study of rock or slag wool workers resulted in an increased risk from nephritis and nephrosis, we think that our pledge for additional data on possible nephrotoxicity of rock or slag wool fibres was justified.

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NOTICE

2nd International Conference on Whole Body Vibration Injuries. November 7-9, 2000. Siena, Italy.

The vibration experienced by some vehicle and machine operators has long been suspected to be a cause of injuries. There are now guides, standards, and proposed legislation intended to protect workers from excessive exposure to whole body vibration and mechanical shock. Seating standards seek to minimise the transmission of vibration and shock to the body. This multidisciplinary conference will provide a unique opportunity to exchange information on the potential for injury from whole body vibration and mechanical shock.

This conference will specially emphasise preventative measures and the promotion of the transfer of knowledge from the laboratory to the field.

The official language of the conference will be English.

The programme will include submitted papers, posters, exhibits, and discussion periods.

Studies to be presented may involve:

- Epidemiology
- Physiological measurements
- Pathological investigations
- Biodynamic measurements
- Models and analogues
- Measurements of exposures
- Seating dynamics
- Other preventative measures
- Guidelines and standards
- Compensation and legal implications
- Review papers

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